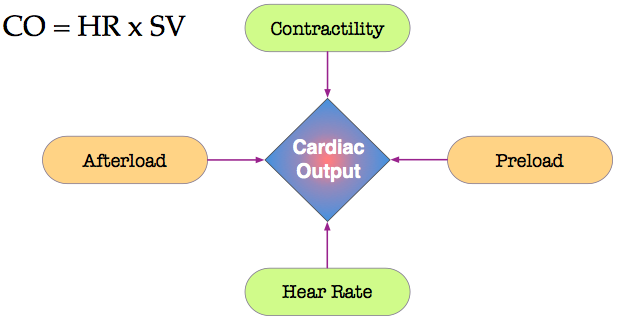
* CO = ΔP \* R =HR \* SV
  + Normal is 5L/min
* CO is governed by the metabolic rate, exercise level, age, and size of the body
  + CO ↑ in response to exercise
  + Obesity ↑ CO b/c adipose tissue is a vascular bed
  + ↑ metabolic activity 🡪 local vasodilation 🡪 ↑ BF to tissue 🡪 CO needs to ↑
* Cardiac Index: CO per m2 of body surface
  + CO ↓ as you age

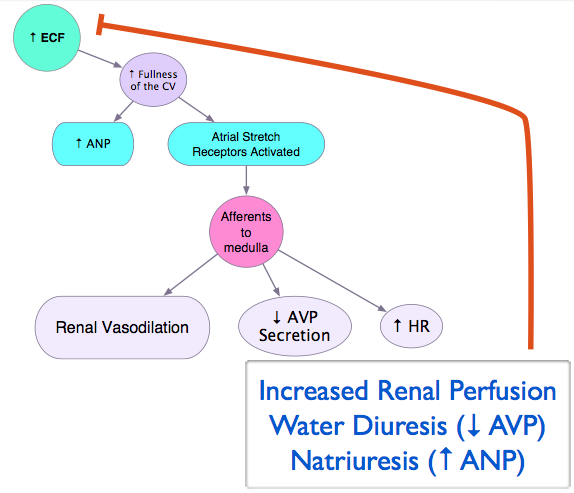


* Contractility and HR are factors of the heart (cardiac determinants of CO)
* Afterload and preload are hemodynamic vascular determinants of CO provided by the vasculature
* HR is the 1st thing to change and strongest response to ↓ in CO
* Preload (VR) is second thing to change
* Contractility is 3rd thing to change
* Metabolism-dependent flow through each vascular bed forms the VR
  + ↑ metabolic activity 🡪 local vasodilation 🡪 ↑ BF to tissue 🡪 CO needs to ↑
* For any given VR/preload 🡪 the cardiac performance is defined as contractility
  + Modulation of contractility, primarily by the ANS, can change CO
* Cardiac hypertrophy does not affect contractility but improves cardiac performance by ↑ myocardial mass

**Long Term Regulation of CO**

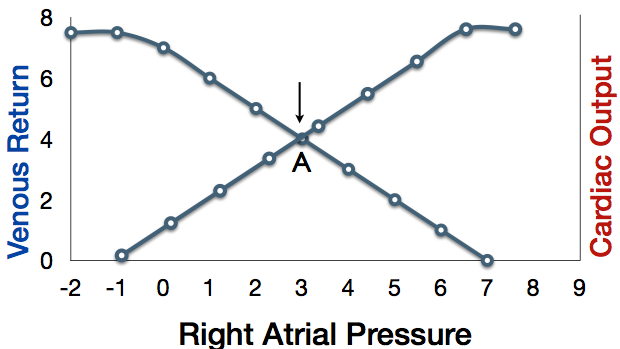
* ↑ in ECF Volume (ECFV) 🡪 ↑ BV 🡪 ↑ VR🡪 ↑ Preload 🡪 ↑ SV 🡪 ↑ CO
* ↓ in ECF Volume (ECFV) 🡪 ↓ BV 🡪 ↓ VR🡪 ↓ Preload 🡪 ↓ SV 🡪 ↓ CO
* \*\*there is no acute regulation of CO

**Sensors and Effectors Regulating BV**

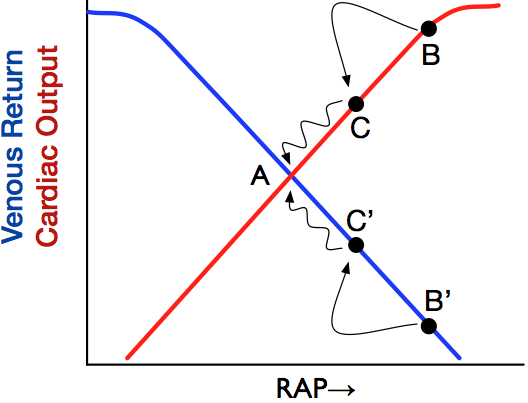
* even if MAP is kept constant
* ****
* Atrial natriuretic peptide (ANP) is released from the Left Atrium in response to ↑ stretch 🡪 induces Na loss from kidneys 🡪 ↓ ECFV 🡪 ↓ BV 🡪 ↓ CO
  + Loss of ANP leads to CHF
  + Natriuresis mean process of excretion of sodium in the urine via action of the kidneys
* Also at ↑ stretch in LA the atrial stretch receptors are activation (these are low pressure baroreceptors—they are not dependent on MAP only on volume) 🡪 activates afferents in the medulla via the vagus n. (PSNS) and activates the NTS 🡪 ↑ HR, ↓ Arginine vasopressin (AVP), & Renal vasodilation (↑ BF to kidneys)
* SO chronically ↑ ECFV will initiate
  + ↑ BV 🡪 ↑ Preload 🡪 ↑ CO 🡪 ↑ MAP
  + compensatory responses:
    - PN & RAS
      * These only respond to Δ in pressure
    - Low Pressure Baroreceptors
      * These respond regardless of ΔP

**Cardiac Function Curves**

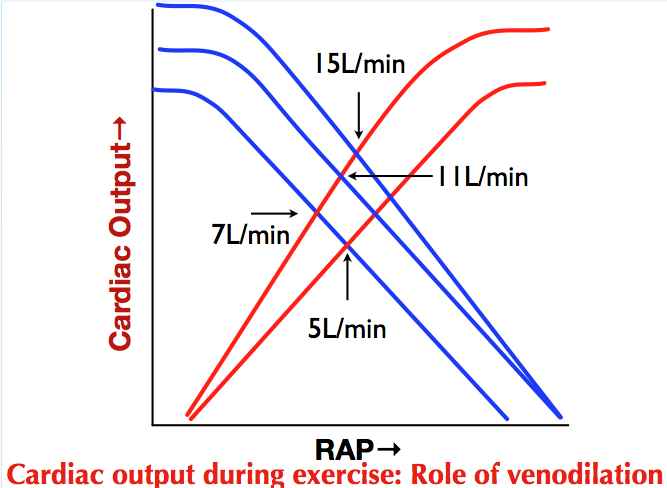
* RAP < CVP
  + If you ↑ RAP then you ↓ VR
* RAP is a commonality between CO and VR
  + At ONE RAP CO=VR
* RAP and VR
  + If you ↑ RAP you ↓ VR and vice versa
  + **VENOconstriction / VASOdilation** (just like blood transfusion)🡪 ↑ Blood going to RA 🡪 so ↑ VR
  + **VENOdilation /VASOconstrction** (just like losing blood) 🡪 more blood stays in veins aand less goes to the heart 🡪 ↓ in VR
  + \*\*remember the P and the blood in the veins is coming from arterioles (which are upstream of veins)—if you ↑ resistance at a vascular bed then upstream you get an ↑ in pressure and downstream a ↓ in pressure) So you if vasoconstrict you will get a ↓ in pressure downstream in the veins which would ↓ VR
* RAP and CO
  + RAP is an INDEX OF PRELOAD so ↑ RAP --↑ in blood in ventricles --↑ CO
  + RAP impedes VR but facilitates CO
  + ↑ **contractility** will ↑ CO
* Cardiac Function Curve

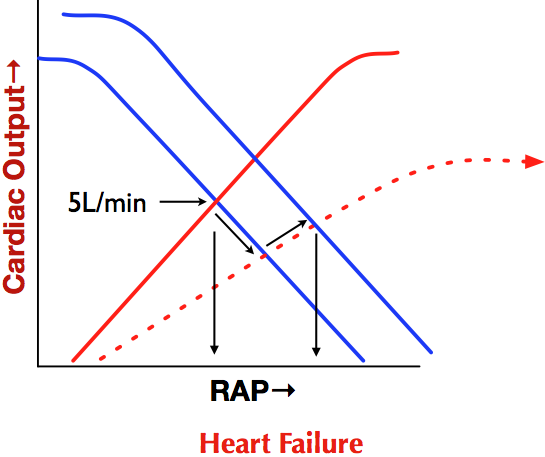


* + At only one value do the two cruves intersect!



* + Cardiac function curves are in equilibrium so if you ↑ RAP then ↑ in CO will result in a ↑ VR so ↓ RAP w/in ~4 beat and it will be back in equilibrium
* The only way to permanently reach a higher or lower set point on cardiac function curve is to change either the cardiac or vascular function curves or both!
  + Change either
    - Vasoconstriction/venodilation
    - Vasodilation/venoconstriction
    - Contractility
      * ↑ in contractility can only ↑ CO so much so to get a bigger ↑ in CO you need to change the vasculature function curve
      * heart by itself (contractility) isn’t enough to ↑ CO a lot



* + Venoconstriction doesn't change the slope
  + Vasoconstriciton changes the slope b/c it has an underlying influence on local metabolic activity!
  + In exercise CO is ↑ than normal b/c muscles need more O2 (and to ↑ O2 either need ↑ extraction or ↑ BF)
* 
  + ↑ contractility is a function of ↑ Ca
  + if you have ↑ Ca post MI the remaining myocytes need ↑ contractility so they get ↑ Ca🡪 these myocytes can start dieing b/c too much Ca is impairing their function
  + Heart failure shifts the curve down and to the R
    - Shifting the curve to the R means ↑ in RAP which leads to edema